New Diagnostic Criteria and Classification of Diabetes—Again?

At the end of the 1970s confusion reigned both with regard to the classification of diabetes and to the appropriate diagnostic test. There was enormous variation in diagnostic cut-off values both for the fasting glucose level and after oral glucose loading. The size of the glucose load varied between 50 g and 100 g or was body weight related. Similarly the types of diabetes were loosely divided into 'juvenile onset' and 'maturity onset', with secondary diabetes, chemical diabetes, borderline diabetes and prediabetes all used in ill-defined ways. In 1979 and 1980 the National Diabetes Data Group (NDDG) in the USA[1] and the World Health Organization (WHO) Second Expert Committee on Diabetes[2] made valiant efforts to create order out of chaos. Types 1 and 2 diabetes, Impaired Glucose Tolerance, Gestational Diabetes and 'other types' took centre stage whilst the 75 g oral glucose tolerance test (OGTT) became the gold standard with fasting and 2-hour values defined. There were nonetheless some differences between WHO and NDDG which were resolved in part by a further WHO report in 1985[3] when Malnutrition Related Diabetes Mellitus (MRDM) was added as a major category.

There has been a growing feeling over the intervening period that further revision was necessary. Whilst discussions were taking place at WHO, independently the American Diabetes Association established an Expert Committee to re-examine diagnostic criteria and classification. It was felt, in particular, that with the burgeoning of pathophysiological knowledge about diabetes, a more aetiology-based system could be used. The WHO Consultation which took place in late 1996 was additionally charged with the more daunting task of making recommendations about the diagnosis and classification of diabetic complications.

The ADA Expert Committee reported in 1997[4] whilst Part I of the WHO Consultation appears in the current issue as a consultation document.[5] There was cross-representation between the groups which has certainly helped achieve some consistency in the recommendations. A new fasting plasma glucose level of 7.0 mmol l^{-1} or above is suggested for the diagnosis of diabetes compared with the previous 7.8 mmol l^{-1}. This is based both on equivalence with the 2-hour value of 11.1 mmol l^{-1} and on predictive power for microvascular complications, albeit in cross-sectional studies. So far so good! The ADA, however, makes a strong recommendation that the fasting plasma glucose (FPG) can be used on its own and that in general the OGTT need not be used. The WHO Consultation by contrast argues strongly for the retention of the OGTT and suggests using the FPG alone when circumstances prevent performance of the OGTT.

The ADA recommendation has already generated considerable steam. It has been shown: (1) that different individuals may be classed as having diabetes using the FPG versus the 2-hour values; and (2) that more people may have diabetes in toto using the ADA FPG test than the 2-hour WHO test. This change of classification is shown in the communications of Kerr *et al*.[6] and Unwin *et al*.[7] also in the present issue.

Several points should be made. First both ADA and WHO state that in asymptomatic subjects the diagnosis of diabetes can be made *only* on the basis of at least two abnormal results. The recommendation for clinical classification by WHO has been and remains the 2-hour OGTT measuring *both* the fasting and the 2-hour values. Only for epidemiological studies does WHO recommend using a single test—2 hours post-glucose load or fasting. Few, if any, of the papers published so far criticising ADA (and by implication WHO) meet the requirements for diagnosis of the individual. In terms of *populations* the new criteria make sense—it does not matter whether individuals change category as it is the total prevalence that matters. Kerr *et al*.[6] in this issue indeed make the mistake of referring to only the 2-hour value in classification, and their 4 subjects called IGT by WHO criteria in fact have diabetes according to both ADA and WHO. Other arguments continue about the fasting versus the 2-hour value.

It is claimed that people may not be fasting properly and certainly this can be the case. Equally, particularly in developing countries, subjects will not necessarily have had adequate carbohydrate intake beforehand and a 'diabetic' 2-hour value may result from undernutrition rather than reflect true diabetes.

There has also been discussion about the measurement of glycated haemoglobin as a diagnostic test. Wiener and Roberts[8] in the present issue show that it has high specificity but low sensitivity and suggest it could be used as an initial screen. This may be useful in rich countries, but until the test becomes better standardised and much cheaper it is unlikely to be of use in much of the world.

The classification is perhaps less controversial. We now have Type 1 and Type 2 diabetes again, moving away from the clinically confusing terms of IDDM and NIDDM. Type 1 refers to the beta cell destructive process found in the usual Northern autoimmune diabetes, but also to the non-autoimmune type found in some non-Europid populations. Impaired Glucose Tolerance (IGT)

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is relegated to a risk category and is joined by Impaired Fasting Glycaemia (IFG; Impaired Fasting Glucose in the ADA version). Other Types include all those where aetiology is more clear, such as the MODYs and the former fibrocalculous pancreatic diabetes variant of MRDM. One major difference remains in Gestational Diabetes Mellitus (GDM). ADA has stuck to its historic testing and criteria whilst WHO includes both IGT and new diabetes in pregnancy under the banner of GDM.

Obviously criticisms are possible of both ADA and WHO. We would like to hear preferably constructive criticisms about the WHO Consultations' proposals before establishing a final version. Thereafter we hope that prospective studies will prevail. We stress that the whole purpose of revising the diagnostic criteria is to help establish those at risk of the specific diabetic complications so that preventive measures can be instituted as soon as possible.

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References

1. National Diabetes Data Group. Classification and diagnosis of diabetes mellitus and other categories of glucose intolerance. *Diabetes* 1979; **28**: 1039–1057.
2. WHO Expert Committee on Diabetes Mellitus. *Second Report*. Geneva: WHO, 1980. Technical Report Series 646.
3. World Health Organization. *Diabetes Mellitus: Report of a WHO Study Group*. Geneva: WHO, 1985. Technical Report Series 727.
4. The Expert Committee on the Diagnosis and Classification of Diabetes Mellitus. Report of the Expert Committee on the diagnosis and classification of diabetes mellitus. *Diabetes Care* 1997; **20**: 1183–1197.
5. Alberti KGMM, Zimmet PZ for the WHO Consultation. Definition, diagnosis and classification of diabetes mellitus and its complications. Part 1: diagnosis and classification of diabetes mellitus. Provisional report of a WHO Consultation. *Diabet Med* 1998; **15**: 539–553.
6. Kerr D, Cavan DA, Everett J. Diabetes—What's in a name? *Diabet Med* 1998; **15**: 619.
7. Unwin N, Alberti KGMM, Bhopal R, Harland J, Watson W, White M. Comparison of the current WHO and new ADA criteria for the diagnosis of diabetes mellitus in three ethnic groups in the UK. *Diabet Med* 1998; **15**: 554–557.
8. Wiener K, Roberts NB. The relative merits of haemoglobin A_{1c} and fasting plasma glucose as first-line diagnostic tests for diabetes mellitus in non-pregnant subjects. *Diabet Med* 1998; **15**: 558–563.